REMARKS

Status of the claims:

Claims 1-7 and 23-25 and 27 are pending. Claims 1-7 and 23-25 and 27 stand rejected. Claim 26 was canceled previously. The claims have been amended as explained below. No new matter has been added.

Claim Rejection under 35 USC § 112:

Claims 1-7 and 23-27 were rejected for lack of enablement for the "prevention" of Alzheimer's and other diseases. Claim 1 has been amended to remove the words "or prevention."

Claim Rejection under 35 USC § 112:

Claims 1-7, 24 and 27 were rejected for lack of enablement for the treatment of the following diseases: mild cognitive impairment Down's syndrome, Hereditary Cerebral Hemorrhage with Amyloidosis of the Dutch-Type, cerebral amyloid angiopathy, other degenerative dementias, dementias of mixed vascular and degenerative origin, dementia associated with Parkinson's disease, dementia associated with progressive supranuclear palsy, and dementia associated with cortical basal degeneration.

Claim 1 has been amended to remove the listed diseases. Claim 24 has been cancelled. Claim 1 spelling of "compriseing" has been corrected to "comprising."

Claim Rejection under 35 USC § 112:

Claims 1-7 and 23-27 were rejected under 103(a) as unpatentable over Göshke (U.S. 5,559,111) in view of Savaskan (Neurobiology of Aging, 22, 541-46, 2001).

Göshke teaches the compounds required by the claims but only as anti-hypertensive agents, as renin-inhibitors, and their administration to human beings, but does not teach or suggest administration for the treatment of Alzheimer's disease.

Savaskan teaches that the mammalian brain contains detectable angiotensin converting enzyme (ACE) and Angiotensin II and their receptors. Savaskan also teaches Angiotensin II inhibits acetylcholine release, and notes that Angiotensin II MAY be related to cholinergic function (page 542, column 1, paragraph 1). That reference also notes that increased ACE activity MAY be responsible for cognitive impairment in AD (page 544, column 2, paragraph 2). The authors conclude that this MAY explain the behavioral eliciting effects of ACE inhibitors on passive avoidance and retention performance [emphasis added].

The same paragraph continues with more conjectures: An additional effect of ACE in cortex MAY be based on neuropeptide

regulation, since ACE is involved in the degradation of several neuropeptides such as bradykinin, encephalins, substance P and neurotensin. The increase in ACE immunoreactivity in AD, which was accompanied by angiotensin II increase in some control, but all AD cases, MAY reflect the enhanced RAS activity in the disease progress [emphasis added].

Savaskan did not state or suggest that the proteolytic kidney enzyme renin, or compounds angiotensinogen or angiotensin I are found in the mammalian brain. Neither has Savaskan suggested that inhibition of the renin enzyme would affect the level of ACE or ACE activity in the mammalian brain. As indicated above, ACE in the cortex could affect numerous neuropeptides, providing only circumstantial evidence of correlation with Angiotensin II with AD.

Based on Göshke in view of Savaskan, there was no reasonable expectation of success. The use of a renin inhibitor for the treatment of Alzheimer's disease is not a known method and would not, in view of the cited references, be expected to yield a predictable result. The skill level or state of the art is low, as noted by the Examiner on page 9 of the office action.

Thus, these references would not have motivated those skilled in the art to explore renin inhibitors for use in treatment of AD since, as noted by the Examiner, "only Acetylcholinesterase inhibitors and NMDA-antagonists" classes

have been effective. A combination of the cited references does not amount to disclosure of treatment of Alzheimer's using a renin inhibitor. It is not a simple matter of substitution to provide a renin inhibitor to reduce ACE activity to give a predictable result of Alzheimer's disease treatment. Even if there is some hint of treatment of AD in the combination of the cited references, that hint does not provide the requisite reasonable expectation of success required under § 103.

For these reasons, the Applicants request reconsideration and withdrawal of the § 103(a) rejection of the claims.

The Applicants invite the Examiner to contact the Applicants' undersigned representative at (312) 913-2136 if the Examiner believes that this would expedite prosecution of this application.

Respectfully submitted,

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